THE MOLECULAR MECHANISMS OF TENDON PATHOLOGY

M. Collins

MRC/UCT Research Unit for Exercise Science and Sports Medicine, Medical Research Council

Although there is a high incidence of tendon pathology as a result of participation in physical activity, the molecular mechanisms responsible for such pathology are poorly understood. Tendons have a highly ordered hierarchical structure made up of tightly packed protein bundles consisting predominately of type I collagen fibres. Trace amounts of other collagens, such as type V, and other extracellular matrix proteins, such as tenascin-C, are also important structural components of tendons. The expression of genes encoding for proteins found in tendons have been shown to be altered in tendon pathology. In addition, we have recently shown that polymorphisms within both the tenascin-C and COL5A1 genes are associated with Achilles tendon pathology.

In tendons, type V collagen forms heterotypic fibres with type I collagen where it is believed to play an important role in regulating fibrillogenesis and modulating fibril growth. The protein might therefore influence the tensile strength of tendons.

Tenascin-C, on the other hand, is able to bind to various components of the extracellular matrix and to cell receptors. These interactions are believed to play an important role in regulating cell-matrix interactions. The expression of the tenascin-C gene is also regulated by mechanical loading. Investigators have suggested that abnormal mechanical loading initiates apoptosis in tendon cells prior to the development of tendinopathy . Since mechanical signals are able to alter the synthesis of tenascin-C, which in turn is able to regulate cell-matrix interactions, this protein may play an important role in the proposed apoptotic model of tendinopathy.

Therefore both type V collagen and tenascin C might be involved in the aetiology of Achilles tendon pathology.